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REVIEW PAPER

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Left ventricular hypertrophy in athletes and hypertensive patients

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Systemic hypertension and physical exercise are both associated with cardiac adaptations. The impact is most prominent on the left side of the heart, which hypertrophies leading to left ventricular hypertrophy. This article reviews structural and functional cardiac changes seen in hypertensive and athlete's hearts.

1 | INTRODUCTION

Chronically or intermittently elevated blood pressure (BP) increases systemic pressure and volume overload, with increased workload on the left ventricle and ultimately left ventricular (LV) hypertrophy (LVH). The normal left ventricle size (Table 1) undergoes several types of anatomical cardiac structural adaptations varying from concentric remodeling, eccentric remodeling, concentric hypertrophy, and eccentric hypertrophy to a combination of concentric and eccentric hypertrophy.[1] Hypertensive LVH is a well-recognized risk factor for heart failure, myocardial infarction, arrhythmias, sudden cardiac death, and stroke.[2–4] Physical activity increases heart rate and BP. Regularly performed sports or physical activities of substantial volume and intensity lead to cardiac changes that meet the characteristic criteria for LVH, especially in highly trained individuals.[5,6] However, cardiac adaptations in response to increased physical activity, referred to as "athlete's heart," differ from the cardiac changes resulting from the pathologically elevated BP in hypertension. The objective of this review is to discuss and contrast the pathological and physiological cardiac adaptations associated with hypertension and chronic exercise.[7,8]

2 | HYPERTENSIVE HEART

Chronic, untreated systemic arterial hypertension leads to end organ damage.[3,4,8] The heart in chronic hypertension responds to the increased hemodynamic load with structural and functional changes. The structural changes include hypertrophy of existing myocytes and addition of sarcomeres together with an increase in connective tissue, ultimately leading to an overall increase in ventricular mass.[3,9,10] LV structural changes can include concentric or eccentric remodeling, concentric or eccentric hypertrophy, or a combination of concentric or eccentric hypertrophy,[1] with a varying combination of increase in LV wall thickness and LV diastolic and systolic dimensions (Table 2). In a recent publication from the Dallas Heart Study where 31% of the population was hypertensive, the prevalence of concentric LVH was much higher than that of eccentric hypertrophy. LVH was present in 730 (30%) of the 2458 patients, classified as indeterminate in 404, isolated thick (concentric) hypertrophy in 289, dilated (eccentric) hypertrophy in 30, and both thick and dilated hypertrophy in seven patients. On follow-up, outcome was worse in patients with dilated (eccentric) hypertrophy than in those with isolated thick (concentric) hypertrophy and worst in those who had a combination (both thick and dilated hypertrophy) of the two types of hypertrophy.[11]

In addition to structural changes, functional compensations with neurohormonal recruitment of sympathetic or renin-angiotensin aldosterone systems also ensue.[12] Initially, these adaptations are necessary in normalizing LV wall stress, preserving LV mechanical function, and ultimately cardiac output against an increasing afterload.[4,8] However, these compensatory mechanisms are deleterious in the long term, leading to diastolic and/or systolic dysfunction. When these cardiac adaptations are no longer sufficient to compensate for the increased ventricular wall stress, myocyte death ensues, cardiac wall thickness decreases, and LV cavity increases, resulting in eccentric LVH. These cardiac changes lead to a progressive decline in contractility and ultimately systolic heart failure.[13] However, even without a decrease in ejection fraction, hypertensive LVH is associated with increased cardiovascular risk and is a robust prognostic marker for adverse cardiovascular events and an independent predictor of sudden cardiac death.[11,12]

The European Society of Hypertension and the European Society of Cardiology as well as the European Association of Cardiovascular Imaging and the American Society of Echocardiography have proposed

TABLE 1 Normal left ventricular indices based on linear and two-dimensional assessments

	Women	Men
Linear method		
Posterior wall thickness, cm	0.6–0.9	0.6–1.0
Septal thickness, cm	0.6–0.9	0.6–1.0
Relative wall thickness, cm	0.22–0.42	0.24–0.42
Left ventricular mass, g	67–162	88–224
Left ventricular mass/BSA, g/m^2	43–95	49–115
Two-dimensional method		
Left ventricular mass, g	66–150	96–200
Left ventricular mass/BSA, g/m^2	44–88	50–102

Abbreviation: BSA, body surface area.
From Lang et al.[1]

recommendations for the assessment of LVH using electrocardiography, echocardiography, and other methods.

Electrocardiography of the heart is the least costly and most widely available method. The Sokolow-Lyon index, Romhilt-Estes score, or Cornell voltage criteria can be utilized for the detection of LVH.[10]

Echocardiography, however, has been the gold standard for assessing LV size and function. LV mass (LVM) is calculated using the LV internal dimension, interventricular septum and LV posterior wall thickness at the end of diastole, American Society of Echocardiography recommendations, or the Penn convention. The cutoff criteria for diagnosing LVH are sex-specific and it is recommended that LVM index be calculated based on the body surface area.[1,14,15]

3 | ATHLETE'S HEART

Cardiac adaptations also occur to accommodate the increased workload observed during exercise; however, there are several distinctions between exercise-induced LV changes compared with hypertensive LVH (Table 3). Exercise-related morphological changes in cardiac structure were first described by Henschen in 1899,[16] who observed enlarged hearts in cross-country skiers–a condition he coined "athlete's heart." Henschen believed the morphological cardiac changes were a normal adjustment to exercise.

However, despite early reports that the increased heart size in athletes had no serious health consequences,[17] several authors have expressed concerns about the long-term consequence of this LV remodeling.[18]

In more recent times, advances in echocardiography have allowed more precise evaluation of the athletic heart. It is now accepted that structural and functional changes, commonly referred to as the athletic heart syndrome or athlete's heart, occur with exercise as an adaptation to the increased physical workload.[19]

The exercise-related cardiac adaptations are highly specific to the type of exercise (Table 4).

The two traditional types of exercise training are aerobic or endurance and anaerobic or strength training. The classic examples of aerobic activities include long-distance running, cycling, or swimming; while resistance or strength training exercise is considered anaerobic. It should be noted that most types of exercise incorporate some elements of both aerobic or endurance and anaerobic or strength training.

Prolonged exposure to purely aerobic training leads to cardiac remodeling characterized by increases in left and right ventricular chamber dimensions and left atrial cavity size and normal systolic and diastolic function. LV wall thickness that exceeds normal upper limits of 13 to 15 mm is also evident in most athletes.[20] Conversely, resistance training alone results in a mild increase in wall thickness, often disproportionate compared with cavity size, but within the accepted normal range, and no changes in LV chamber size. Some misunderstanding persists as to whether strength or resistance training alone results in concentric LVH.[19] However, absolute values uncorrected for body surface area usually remain within the accepted range of normal (Table 1).

Sports comprised of both aerobic and anaerobic types of activities (prolonged cycling, rowing, and swimming) lead to structural and functional cardiac adaptations that reflect the combined demands of the particular sport or activity. These athletes have the most extreme increases in both LVM.[21] It is important to emphasize that an increase in either alone (wall thickness or LVDD) are not physiologically desirable. LV dilatation without comparable increase in wall thickness leads to an inappropriate increase in wall tension that is detrimental to the heart.[6,22]

In general, chronic cardiac adaptations resulting from vigorous, chronic exercise as seen in athletes are considered normal physiologic responses to the hemodynamic demand of the particular sport or physical activity. They are not associated with diastolic dysfunction, arrhythmias, or adverse prognosis, manifestations observed in

TABLE 2 Abnormal left ventricular wall thickness and mass by linear method

	Men			Women		
Abnormality	Mild	Moderate	Severe	Mild	Moderate	Severe
Posterior wall thickness, cm	1.1–1.3	1.4–1.6	>1.6	1.0–1.2	1.3–1.5	>1.5
Septal thickness, cm	1.1–1.3	1.4–1.6	>1.6	1.0–1.2	1.3–1.5	>1.5
Left ventricular mass, g	225–258	259–292	>292	163–186	187–210	>210
Left ventricular mass/BSA, g/m^2	116–131	132–148	>148	96–108	109–121	>121

From Lang et al.[1]

TABLE 3 Hypertensive versus athlete's heart

	Hypertensive heart	Athlete's heart
LV wall thickness	Increased	Increased
LV diastolic dimension	Decreased, normal, or increased	Increased
LV systolic dimension	Decreased, normal, or increased	Increased
Stroke volume	Increased	Increased
Fractional shortening	High, preserved, or depressed	Preserved
Diastolic dysfunction	Often present	Absent
LV wall strain	Present	Absent
Heart rate	Not affected	Bradycardia

Abbreviation: LV, left ventricular.

hypertension-induced LVH,[8,23] and regress quickly when training is discontinued.[7]

However, it is likely that extreme demands on the cardiovascular system such as those imposed by competitive sports (eg, basketball and soccer) may in some cases perpetuate cardiac maladaptations. The American College of Sports Medicine and the American Heart Association recommend low- to moderate-intensity aerobic exercise (brisk walk) of approximately 30 minutes per day most if not all days of the week and should be encouraged by healthcare providers.[24,25] Such exercise is safe for almost all ages and populations with comorbidities and has been shown to have a favorable effect on traditional and novel cardiovascular risk factors,[26] likely leading to LVH regression.[27]

A distinction must also be made between the purely athletic heart syndrome and the changes that occur in hypertrophic cardiomyopathy that can also occur in athletes. This is a pathologic condition seen in patients, who could also be athletes, with primary myocardial disease or significant valvular heart disease. The structural cardiac changes in these individuals are usually much greater than those induced by exercise only. The distinction between these two conditions, true athlete's heart versus structural changes resulting from heart disease, is crucial because the risk for sudden death in young athletes increases when structural heart changes are the result of myocardial or valvular diseases.[28,29]

The emerging concept is that a hemodynamic load threshold exists beyond which the cardiac muscle will make the necessary adaptations to accommodate the increased demand. This hemodynamic load threshold is reflected by a systolic BP of approximately ≥150 mm Hg, as suggested by our findings.[30,31] The level of physical activity that will elicit such response is relative to the individual's peak exercise capacity. For example, according to our findings, the systolic BP ≥150 mm Hg necessary to trigger cardiac remodeling was achieved by relatively low-fit individuals at the workload of 4 to 5 metabolic equivalents (METs). This level of physical activity typically represents approximately 60% of the peak exercise capacity of sedentary or relatively low-fit individuals (estimated peak exercise capacity 6–7 METs). If we assume that 60% of the peak workload is necessary to elicit a systolic BP response ≥150 mm Hg, this workload for a relatively fit individual (estimated peak exercise capacity 12 METs) is 7.2 METs and for athletes (estimated peak exercise capacity 20 METs) is 12 METs. Thus, for relatively fit individuals and athletes, the workload of daily activities (4–5 METs) is not likely to elicit a systolic BP response ≥150 mm Hg necessary to elicit cardiac remodeling. However, such BP threshold is reached and well exceeded during the highly demanding exercise training endured by athletes and, therefore, cardiac remodeling to accommodate the imposed demand is triggered. Nevertheless, the morphologic pattern of physiologic cardiac remodeling induced by athletic conditioning and its differentiation from primary pathologic hypertrophy has become a particularly relevant clinical issue.[32] From the original pathologic descriptions of myocardial hypertrophy in trained individuals from Kirch and Linzbach,[33] two concepts still merit consideration: (1) that the heart of the trained athlete can be twice the normal size but the histologic structure remains intact; and (2) that the weight of the trained heart does not usually surpass the limit of 500 g.

TABLE 4 Differences in LVDD and wall thickness in 947 athletes versus controls

Sport	LVDD, cm	Wall thickness, cm
Cross country skiing	5.41	0.98
Pentathlon	4.35	0.98
Soccer	3.11	0.76
Cycling	5.91	2.02
Swimming	4.9	1.71
Canoeing	4.23	1.71
Rowing	3.87	2.13
Weight training	1.32	1.23
Long-distance track	3.47	1.49
Tennis	2.69	1.0
Boxing	2.25	0.94
Taekwondo (karate)	2.07	0.23
Water polo	2.02	1.38
Volleyball	1.43	0.39
Wrestling/judo	1.25	1.21

Adapted from Spirito et al.[38]

Evidence from the large number of echocardiography studies has described LV morphologic changes in trained athletes. The LV remodeling observed in athletes is considered to be morphologic adaptation to intensive and chronic hemodynamic overload. In fact, there is a sustainable increase in cardiac output owning to reduced afterload and greatly increased preload.[34]

Previous echocardiography studies have shown that LV wall thickness increases by 15% and cavity dimension by 10% with moderate increase in activity in both sexes. Among all trained athletes, LV dimensions are more likely to be substantially enlarged and in a range morphologically compatible with primary cardiac disease. Authors agree that maximal wall thickness in elite male athletes can be 15 to 16 mm.[35] Evidence suggests that in highly trained athletes the distribution of LV wall thickening is quite regular. The most commonly

thickened region is usually the anterior portion of the ventricular septum. Although the different regions of the LV wall may not be thickened to an identical degree, differences between contiguous segments are generally small and therefore the overall pattern of LVH appears symmetric and homogeneous.[36]

Morphologic adaptation to training in athletes enlarges the LV cavity size to an end-diastolic diameter of ≥55 mm. The LV cavity maintains the normal ellipsoid shape, with the mitral valve normally positioned within the cavity and no evidence of LV outflow tract obstruction. The rapid filling phase, or early diastole, is significantly prolonged and associated with decreased rate and volume of filling compared with that of a normal heart.[37]

4 | FITNESS AND LVH

As stated, the exercise BP response at workloads of 4 to 5 METs is attenuated by increased fitness or exercise capacity. Accordingly, in relatively fit individuals, the exercise systolic BP threshold of ≥150 mm Hg necessary to trigger cardiac remodeling is achieved at workloads substantially higher than the workload of 4 to 5 METs observed in relatively low-fit individuals. Since the workload of approximately 4 to 5 METs is similar to the workload of most daily activities, it is reasonable to assume that relatively fit individuals are less likely to exceed the BP threshold of ≥150 mm Hg during daily activities and less likely to develop LVH. This is supported by our findings of an inverse association between exercise capacity, BP response to exercise, and LVM.[27] Furthermore, the systolic BP of physically fit individuals at an exercise intensity of approximately 4 to 5 METs was significantly higher for the low-fit (155±14 mm Hg) compared with the moderate-fit (146±10 mm Hg) and high-fit (144±10 mm Hg) individuals. Similarly, low-fit individuals had significantly higher LVM index (48±12 g/m$^{2.7}$) compared with moderate-fit (41±10 g/m$^{2.7}$) and high-fit (41±9 g/m$^{2.7}$) individuals. In addition, for every 1-MET increase in the workload achieved, there was a 42% reduction in the risk for LVH.[31] Finally, in a randomized controlled study, 16 weeks of aerobic training resulted in significantly lower BP at the exercise intensity of approximately 3 and 5 METs [31] and a significant regression in LVM.[27]

5 | CONCLUSIONS

Even though both hypertensive patients and athletes can develop LVH, there are several structural and physiological differences between the two. That the LV hypertrophies are a function of chronically or intermittently elevated BP is suggested by the aforementioned findings, which show that both fit hypertensive patients and relatively fit individuals are not likely to achieve systolic BP ≥150 mm Hg necessary to stimulate cardiac remodeling during normal daily activities and, therefore, an increase in LVM is not likely. For those with existing LVH, regularly performed aerobic exercise of moderate intensity improves fitness and lowers BP at absolute workloads and the daily hemodynamic load, as is reflected by lower BP. Consequently, the daily exposure to a substantially lower hemodynamic load removes the impetus for cardiac remodeling and eventually leads to LVM regression.

CONFLICT OF INTEREST

The authors of this manuscript have no conflicts of interest to disclose.

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